
miR-24 inhibits apoptosis and represses Bim in mouse cardiomyocytes.

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Public Summary:

Acute myocardial infarction (MI) involves loss of heart cells. One strategy to salvage dying heart cells is to modulate gene expression to promote cell survival without disturbing normal heart function. microRNAs have emerged as powerful regulators of multiple cellular processes, including programmed cell death, suggesting that regulation of microRNA function could serve a cardioprotective function. Here, we report that microRNA-24 (miR-24) expression is downregulated in the ischemic border zone of the murine heart after MI. miR-24 suppresses heart cell death, in part by direct repression of a protein Bim, which positively regulates cell death. In vivo expression of miR-24 in a mouse MI model inhibited heart cell death, attenuated infarct size, and reduced cardiac dysfunction. This anti-cell death effect on heart cells in vivo was partially mediated by Bim. Our results suggest manipulating microRNA levels during stress-induced cell death may be a novel therapeutic strategy for heart disease.

Scientific Abstract:

Acute myocardial infarction (MI) involves necrotic and apoptotic loss of cardiomyocytes. One strategy to salvage ischemic cardiomyocytes is to modulate gene expression to promote cell survival without disturbing normal cardiac function. MicroRNAs (miRNAs) have emerged as powerful regulators of multiple cellular processes, including apoptosis, suggesting that regulation of miRNA function could serve a cardioprotective function. In this study, we report that miR-24 (miRNA-24) expression is down-regulated in the ischemic border zone of the murine left ventricle after MI. miR-24 suppresses cardiomyocyte apoptosis, in part by direct repression of the BH3-only domain-containing protein Bim, which positively regulates apoptosis. In vivo expression of miR-24 in a mouse MI model inhibited cardiomyocyte apoptosis, attenuated infarct size, and reduced cardiac dysfunction. This antiapoptotic effect on cardiomyocytes in vivo was partially mediated by Bim. Our results suggest that manipulating miRNA levels during stress-induced apoptosis may be a novel therapeutic strategy for cardiac disease.

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